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AMERICAN ASSOCIATION OF INHALATION THERAPISTS

"Inhalation Therapy"

"Inhalation Therapy" is the official publication of the American Association of Inhalation Therapists, an organization of therapy technicians working in hospitals, for firms providing emergency therapy service and for municipal organizations. The Association is sponsored jointly by the American College of Chest Physicians and the American Society of Anesthesiologists. Contents include news and information pertinent to the profession including medical research, operative techniques, and practical administration.

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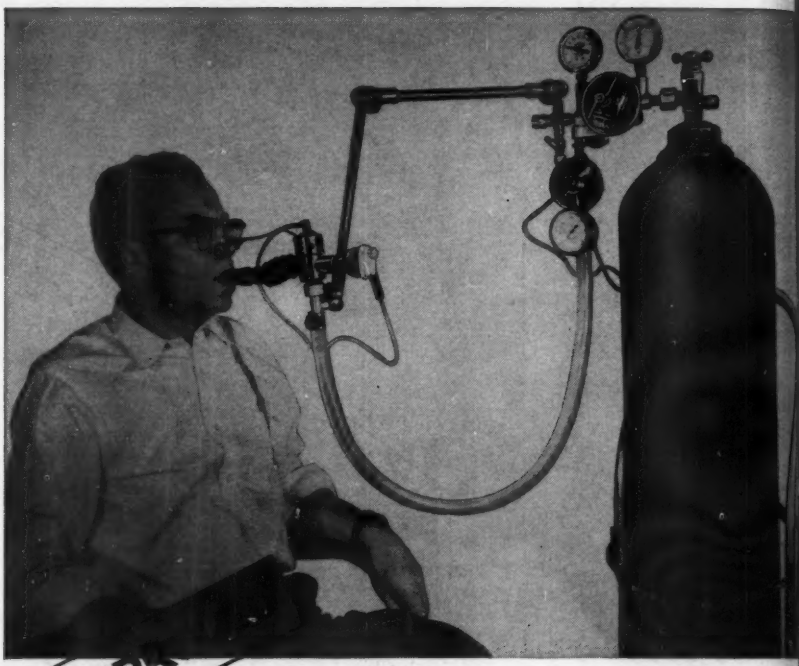

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Editorial

"May Your Offspring Be Numerous, Healthy, Wealthy and Wise"

THE above is a traditional Eastern European wedding wish. As such, it is perhaps peculiarly fitting at this time, for, as you already know, a wedding of sorts has just been consummated between the American College of Chest Physicians and the American Society of Anesthesiologists. To carry the simile still further, the progeny comprises the American Association of Inhalation Therapists.

As indicated in the wedding wish, it is the offspring which engage the hopes and aspirations of the happy parents and all their friends. Here, too, all the work and thought that has gone into arranging for this dual sponsorship for the AAIT has been done with only one aim in view.

It is our feeling that the Inhalation Therapy technicians in this country, at best, are on a par with the most highly informed group of medical technicians in the profession. As such, they warrant considerably more respect and dignity than they now command.

Unfortunately, country-wide, they are not always represented by the best.

Elsewhere in this issue can be found the new, combined Advisory Committee of the American Association of Inhalation Therapists, representing the two sponsoring groups. (See pages 5 and 11.)

We feel strongly that two major efforts must be made. Your society must be made strong, both numerically and organization-wise. This is a pre-requisite for success, since the contribution of many individuals working *together* will be greater than that of the same individuals working independently.

The second effort must be devoted toward raising the standards of education, training and background of Inhalation Therapy technicians. This must be accompanied by some form of accreditation and registry, so that the chaff can be separated from the wheat.

The Advisory Committee has already, through the American Medical Association, made an important start towards accomplishing the second effort.

The first is up to you. YOU must see that the AAIT gets more members. YOU must see that you attend all meetings. YOU must participate in discussions, elections and policy-making, so that the organization truly represents your needs and desires.

Your Advisory Committee is for YOU. We offer you the best that we can do to further your ambitions and desires. Help us make these dreams come true so that—together we may think of the wedding wish and say a fervent AMEN.

Edwin Emma, M.D.

At AAIT Annual Meeting —

Lecture Series to Emphasize Emergency Inhalation Therapy

INHALATION therapy in times of catastrophe and emergency will be the topic for a full day of talks and panel discussions at the forthcoming annual meeting and lecture series of the American Association of Inhalation Therapists. The meeting is to be held in the Hotel Hollenden in Cleveland, Ohio, November 4 through 8. Panel discussions on topics of importance to inhalation therapy, moderated by doctors well-known for their work in the field, will highlight each day's activities.

A panel discussion Wednesday, November 6, on "Inhalation Therapy in Catastrophe" will be led by Dr. R. A. Hingson of Western Reserve University Hospitals. To supplement his talk and discussion, Dr. Hingson has invited those attending the meeting and lecture series to take a field trip to his hospital Wednesday evening. A very moving demonstration of resuscitation and inhalation therapy in the hospital amphitheater is planned.

Dr. Edwin Levine of Edgewater Hospital in Chicago will be the moderator of panel discussion on Pressure Breathing on Tuesday, November 5. On Thursday, November 7, a panel discussion on the general subject of Oxygen Tent Therapy will be moderated by Dr. Albert Andrews of St. Luke's Hospital, Chicago, and on Friday, Dr. Meyer Saklad of Rhode Island Hospital, Providence, Rhode Island, will be the moderator of a panel discussion covering the organization and maintenance of an inhalation therapy department.

Dr. Saklad's panel is designed to cover the entire field of inhalation therapy and his panel members will represent hospitals, service companies and municipal organizations. Russell C. Nye, Administrator of Northwestern Hospital, Minneapolis, will present the hospitals' point of view, Col. Joseph McCarthy of the Chicago Fire Department will cover the municipal organizations, and J. H. Newell of the American Medical Oxygen Service, Hammond and Gary, Ind., is scheduled to represent the service companies.

Commercial exhibits will be on display Tuesday, Wednesday and Thursday during the week of the meeting, and on Thursday evening the exhibitors will have a program of educational films for those attending the convention.

Luncheon sessions will be held each day and the luncheon programs have been planned to show how inhalation therapy, as a relatively new paramedical specialty, fits into the overall medical scene. Such topics as "Current Research in Inhalation Therapy" and "Inhalation Therapy and the Practice of Medicine" will be covered.

In addition to the panel discussions, which are to be held from

10:30 to 12:00 each day, scientific and technical discussions by doctors prominent in the field of inhalation therapy are scheduled. "Physiologic Basis of Inhalation Therapy" will be discussed by Dr. H. F. Helmholz, Jr., of the Mayo Clinic; Dr. David Gillespie, Cleveland City Hospital, will talk on "Intermittent Positive Pressure Breathing"; Dr. Joseph Miller of Mobile, Alabama, "Aerosol Therapy"; Dr. Vincent Collins of St. Vincent's Hospital, New York City, "Cardiac, postoperative and convalescent"; Dr. Paul Searles, Presbyterian-St. Luke's Hospital, Chicago, "Masks, Tents and Catheters"; Dr. J. M. Brown, Medical College of South Carolina, "Advances in Polio Treatment"; Dr. B. B. Sankey, St. Luke's Hospital, Cleveland, "Gases Other than Oxygen"; Dr. R. I. Shapiro, Detroit, "Qualifications and Standards for Inhalation Therapists."

Members of the American Association of Inhalation Therapists will pay a registration fee of $40.00 to attend the meeting and lecture series and non-members will pay $50.00. The registration fee will cover the cost of five luncheons, as well as admission to all lectures, the field trip and exhibits.

Members will receive a printed program with their next news letter.

For additional data, please write to:

Albert Carriere
Executive Director
American Association
of Inhalation Therapists
332 South Michigan
Chicago 4, Illinois

WHAT TO DO IN EMERGENCIES

— *How the Chicago Fire Department Handles Them*

The Chicago Fire Department began handling emergency ambulance calls about 1928, with a few rebuilt trucks for ambulances, and using the old time pulmotors for resuscitation of drowning and asphyxiation victims. In 1946, Joseph J. McCarthy was asked to develop an extensive expansion and modernization program. During the past 11 years, much progress has been made in the organization, equipping and training of the department.

Under the direction of Fire Commissioner Robert J. Quinn, Mr. McCarthy supervises the Emergency Ambulance Service, and the training program for the firemen who serve on it.

This service provides coverage of four types: inhalator calls, street accidents, fires and catastrophes. The area served comprises some 213 square miles in Chicago and about 10 square miles at O'Hare airport. Distribution of units, dispatched by 2-way radio, is such

Chicago fireman, who is a member of the Ambulance Service demonstrates how inhalation and resuscitation can be carried on while transporting an emergency case.

that any part of the city may be reached within two minutes.

16,000 Calls a Year

The emergency service now averages between 16 and 17 thousand calls annually for inhalators alone. These calls are taken by 16 ambulances and 13 rescue squads. The program is still expanding and 5 more ambulance units will be equipped and in operation by early 1958. Out of some 14 thousand calls in the past six months, over 9 thousand were for inhalator service. This is for people who have had heart attacks, drowned or been overcome by gas or smoke.

Other emergency calls include cases of childbirth, accidents of all sorts from falling down cellar stairs to having part of a building collapse on the victim, standby calls to airports for landings of planes either burning or known to be in trouble which might result in crash landing, etc.

The emergency service cooperates very closely with the civil defense authorities, and Chicago boasts the only fire department in the world that has complete first aid equipment in every station, including all fire boats.

Dr. Harold P. Sullivan advises Mr. McCarthy in all medical matters. Recently it was decided on the basis of research and current medical opinion to switch to the use of 100% oxygen for the resuscitators. The Fire Department had previously used a mixture of 93% oxygen and 7% carbon dioxide, which was a common practice of rescue squads in many places until comparatively recently.

All Firemen Trained

All firemen – even those not on emergency ambulance and rescue squads – receive first aid training and a 4-hour course in the use of the E&J resuscitator, which is standard equipment carried by the ambulance service. This training is reviewed and brought up to date every other year. Some 4300 men have been retrained during the past four months.

To join the emergency service, a fireman has to have completed 10 years of service in the fire department, and must be a qualified

Interior of Chicago Ambulance Service unit. In major emergency litters can be suspended from ceiling to carry four persons.

driver and of high moral calibre. Aptitude for this sort of work must also be demonstrated, since not everyone who says he is interested in emergency work can actually give the high quality performance required, according to McCarthy.

Training is done in a special school and in the field. Each candidate must pass a 15-hour Instructor's Course in First Aid conducted by the Red Cross. That is, each man must not only be able to give first aid himself; he must also be qualified to teach it to others. This is followed by another 15 hours of advanced first aid and rescue techniques, demonstrations, visual aids, etc. The men are taught to use the Nielson method of artificial respiration wherever use of the resuscitator is not feasible. They are taught to "splint fractures where they lie," and to control bleeding and breathing.

McCarthy's men stay with the victim until adequate medical care is present – either by arrival of a physician who takes over, or by taking the victim to the nearest hospital themselves. In the latter case, first aid and/or resuscitation is continued in the ambulance en route to the hospital.

CO-SPONSORSHIP BY ANESTHESIOLOGISTS NOW OFFICIAL

The AAIT is now officially co-sponsored by the American Society of Anesthesiologists and the American College of Chest Physicians. The ASA has appointed the following doctors to serve on AAIT's Advisory Board.

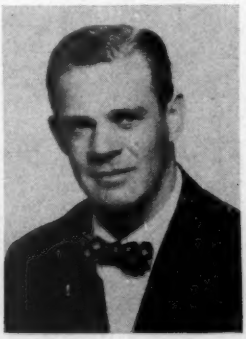
Dr. Meyer Saklad, Director of the American Board of Anesthesiology and of the ASA, is Chief of the Dept. of Anesthesiology of Rhode Island Hospital in Providence.

Dr. Vincent J. Collins, new President of the N. Y. State Society of Anesthesiologists, and Secretary of AMA's Section on Anesthesiology, is Director of the Dept. of Anesthesiology at St. Vincent's Hospital in New York City.

Dr. Edwin Emma, Diplomate of the American Board of Anesthesiologists, and Attending Anesthetist at Gouverneur Hospital, is Director of Anesthesia at Booth Memorial Hospital, Flushing, N. Y.

Meyer Saklad, MD

Vincent Collins, MD



Edwin Emma, MD

Respiration Physiology

The Control of Respiration

WHEN we want to, we can deliberately breathe rapidly or slowly, deeply or shallowly, or we can hold our breath—up to certain limits. Also, we can modify the exhalations in speech or singing, etc. But what about the times when we are not thinking about our breathing? Probably 90% of the time we are not aware of it at all; and at other times when we are conscious of it—such as when we hurry up stairs and notice shortness of breath—we are still not exerting any voluntary control over it. So it is evident that most of the regulation is unconscious, and further, that it is automatically adjusted to meet changing circumstances.

This is the third in a series of articles on basic respiration physiology. The December issue will carry the final one, in which the transport of respiratory gases by the blood will be described.

This regulation of the respiratory processes is effected by both nervous and chemical mechanisms.

Innervation

It will be recalled that the respiratory movements are accomplished by the diaphragm and the two sets of intercostal muscles—the "accessory" muscles of head, neck, chest and abdomen ordinarily being used only in vigorous exercise. These muscles are supplied with nerve fibers, some of which carry impulses to the brain; others relay them from the brain to the muscles. The nerves to the diaphragm are called the *Phrenic* nerves, one to each side; those supplying the intercostal muscles bear the same name.

There are also fibers conveying messages from the lung tissue itself to the brain. These fibers terminate in receptor organs which are sensitive to physical distortion—specifically to stretching. Hence, as the lung tissue in which they are embedded expands, the receptors are stretched and this causes them to send impulses along the *Vagus* nerve to the brain.

The Respiratory Center

There are three nerve centers of the brain which are involved with respiratory regulations. These are named the *Pneumotaxic Center* (located in the Pons), and the *Expiratory* and *Inspiratory* centers (both in the Medulla). They are all usually spoken of collectively simply as the Respiratory Center. It will be realized, of course, that

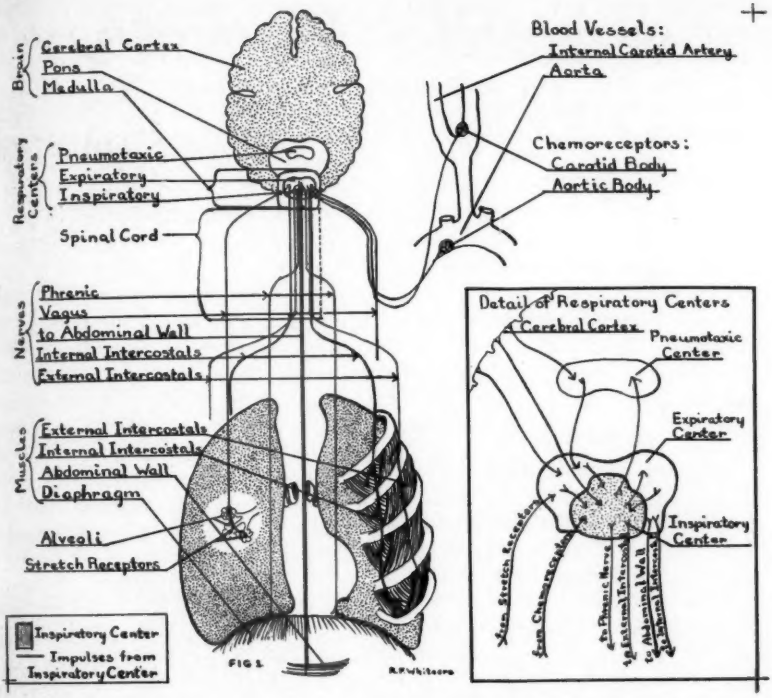


Fig. 1.—Schematic diagram to illustrate the sources and inter-relation of nervous and chemical influences upon the respiratory center, as well as the pathways by which it exerts control over pulmonary ventilation.

higher centers of the brain are also capable of influencing respiratory activity (as in the case of voluntary control, emotional states, reactions to pain, etc.); but these are all mediated through the Respiratory Center. Hence, the latter receives signals from a number of sources both within the respiratory machinery and outside it. Its three sections are all connected with each other to form a complex intercommunication system. The orders it sends out along the phrenic and intercostal nerve fibers are the results of decision it makes on the basis of this intelligence.

Figure 1 is a schematic diagram of the connections between the respiratory center and points from which it receives or to which it sends messages. The Inspiratory Center and all nerves carrying impulses from it are shown in red; all other structures and pathways are black.

Investigations[1, 3, 5] have demonstrated that the Inspiratory Center is really the dominant member of the trio, and that if unopposed, it causes the respiratory musculature to stop in the maximum inspiratory position. However, the impulses impinging on it

from the other two sections of the center (which in turn are being acted on by the cerebral cortex, the vagus, etc.) bring about periodic restraint of its activity, so that inspiration is released and gives way to expiration.

Each of these centers is sensitive to the signals it receives, and the more signals there are, the more excited the center becomes. Correspondingly, the more aroused the nerve center is by increasing numbers of incoming calls, the greater the number of orders it initiates.

Normal Cycle

Now let us trace the events of a normal respiratory cycle. The Inspiratory Center sends impulses (red lines in Fig. 1) via phrenic nerves to the diaphragm and via external intercostal nerves to those muscles, which then contract, raising the ribs upwards and outwards and lowering the diaphragm. As the thorax is thus made larger, the lungs expand and follow its wall, by virtue of the negative intrapleural pressure previously described.

The distension of the lungs stimulates the stretch receptors there and they begin to send impulses up the Vagus to advise the respiratory center that the lungs are filling up. The fuller they get, the more the Vagus prods the Expiratory Center.

Simultaneously with the above process, the Inspiratory Center is sending impulses to the Pneumotaxic Center, and the longer the inspiratory process continues, the more and closer together these impulses to the Pneumotaxic Center. The latter becomes increasingly annoyed by these, and as a result sends more and more impulses down to the Expiratory Center.

The Expiratory Center has by this time become thoroughly aroused by the advices from all quarters, and begins therefore to send inhibitory signals to the Inspiratory Center.

It will be recalled that we have "passive" and "active" types of expiration, the former being the one we use at rest, and consisting merely of a relaxation of inspiratory activity, letting gravity and elasticity of the thorax return the lungs to their normal pre-inspiratory position. In this case, all that is required is a cessation of inspiration, and the Expiratory Center merely inhibits the Inspiratory Center from sending any impulses.

Rapid Evacuation

In situations demanding more rapid and extensive evacuation of the lungs, however, the Expiratory Center, in addition to inhibiting the Inspiratory Center, also sends orders directly to the internal intercostal muscles and abdominal muscles resulting in a forceful expiratory act.

Expiration having thus been initiated, the stretch receptors of the lung are no longer stimulated, and hence stop sending messages up the Vagus. Since the Inspiratory Center has been held temporarily in abeyance during expiration, it has stopped sending remarks to the Pneumotaxic Center, which therefore stops its running commentary to the Expiratory Center (having for the time being nothing further to relay). The latter, since it is no longer being harrassed by complaints from the Vagus and Pneu-

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motaxic Center, desists from inhibiting the Inspiratory Center and stimulating the internal intercostals and abdominals.

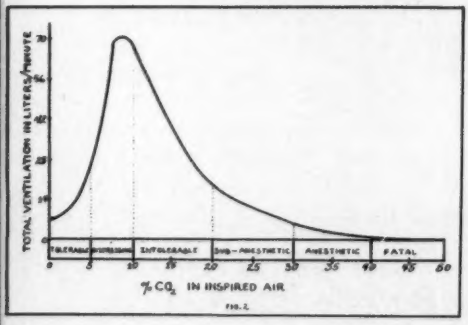
Just as soon as the Expiratory Center relaxes its restraint, the Inspiratory Center discharges another volley of impulses to the inspiratory musculature, and the cycle begins anew.

Complicated as the foregoing may seem, it is in reality a simplification of what really goes on in the Respiratory Center. Suppose, e.g., a foreign body gets lodged in the trachea. Sensory nerve endings there immediately apprise the respiratory center of the need for removing the offending particle before it gets deeper into the lungs. The result is a cough or series of them. Or take, for example, the familiar case of stepping into a cold shower. The quick inspiratory gasp is not because of any real need for air, but because the sudden sensation of cold stimulates the inspiratory center.

When we are excited by fear, anger, or pain our respirations are modified by the effects of these events on the respiratory center.

But we have only begun the long recital of factors influencing the decisions made by the respiratory center, and, indeed, the scope of this article precludes discussion of many. However, in addition to the numerous nervous impulses affecting the center, there are chemical changes occurring in various parts of the body which change the composition of the blood passing through the brain. The nerve cells of the brain are extremely sensitive to such fluctuations, since life as we know it depends on strict adherence to a given set of chemical conditions.

Fig. 2—Effect of CO_2 on Ventilatory Response. Modified from Gray, Pulmonary Ventilation & Its Physiological Regulation, 1950.



Chemical Control

While accumulation of the waste product, CO_2, is offensive to any cell of the body, the cells of the respiratory center are especially intolerant of its presence. Since the blood perfusing them has come from other parts of the body, the Center is thus advised by this chemical messenger that things aren't what they might be, and therefore it institutes measures designed to correct the fault.

Figure 2 shows the ventilatory response to CO_2 in graphic form. The maximum increase is obtained at about 10% CO_2, and beyond that the response falls off progressively as concentration increases. Note especially that any concentration over 5% is distressing to the normal individual, and those over 10% are definitely harmful. However, quite a good increase in ventilation can usually be accomplished by concentrations of 5% or less.

The fact is, of all the known natural stimuli to the respiratory

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center—nervous or otherwise—elevated pCO_2 is the most potent of all, a rise in CO_2 concentration in the *alveolar* air of only 0.2% (corresponds to about 1.5 mm Hg increase in pCO_2 in alveolar air and arterial blood) being sufficient to *double* pulmonary ventilation.[1] And, on the other hand, a lowered pO_2, which one might expect to be a logical stimulus to the respiratory center, actually *depresses* its activity, decreasing ventilation and of course thereby increasing the degree of hypoxia (i.e., making pO_2 even lower).

Right here is where we are brought to the realization that the Respiratory Center is not primarily concerned with oxygen procurement at all: *it is trying to get rid of CO_2!* Getting O_2 *in* is incidental, and of course very desirable, but our metabolism is so arranged that the immediate removal of CO_2 is even more pressing than getting oxygen in.

Take a look again at the gas concentration figures for the respired air. Inspired air contains 20% O_2 and only 0.05% CO_2. Expired air has 15 or 16% O_2 still left in it, but 5% CO_2! If we were in desperate need of oxygen, we would in all probability make more efficient use of the inspired air than this; conversely, it can readily be appreciated that with CO_2 concentrations a *hundred* times greater in expired than in inspired air, any interference with the disposal of this excess CO_2 will result in accumulation of deleterious amounts in the body.

So our ventilatory regulation is directed first and foremost at CO_2 removal, and the O_2 intake is a sort of automatic dividend simultaneously accomplished by the same process. We are killing two birds with one stone, but the bird the respiratory center is most interested in is CO_2 elimination.

Chemo-Receptors

However, there is a different system which *is* interested in oxygen intake. Situated in the arch of the aorta and in the walls of the carotid arteries there are organs called *chemoreceptors* (see Fig. 1 again), which are stimulated by changes in chemical composition of the blood passing through these arteries, just as the respiratory center is. But the chemoreceptors are relatively insensitive to changes in pCO_2, and *are* stimulated by decreases in arterial oxygen tension (low pO_2).

The normal oxygen tension of arterial blood is about 100 mm Hg; that of venous blood (returning from tissues, where oxygen has been taken out) is about 40 mm. The arterial pO_2 must be lowered to 80 or even 70 mm before the chemoreceptors are actuated.[1, 2] Their response consists of sending impulses to the Inspiratory Center to increase ventilation. Oxygen tensions less than 70 mm Hg will of course cause greater stimulation of the chemoreceptors, with a correspondingly greater ventilatory response.

The carotid and aortic body chemoreceptor mechanism is used only in emergencies, however, because in the normal course of events the stimulus of accumulating CO_2 to the respiratory center results in sufficient ventilation to assure good oxygenation. It is only in the case of interference with

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the alveolar oxygenation, resulting in lowered arterial oxygen tension, that the chemoreceptors would come into play. Such a situation develops where there is not enough O_2 in the inspired air (as at altitude, or in atmospheres containing less than 20% O_2) or where disease processes in the lungs are preventing adequate uptake of oxygen by the blood.

A simple experiment that illustrates the relative potencies of low oxygen and high carbon dioxide concentrations as respiratory stimulants is as follows: Three spirometers are set up for the subject to breathe from. The first contains room air and has incorporated in its circuit a canister of soda lime which absorbs CO_2 in the expired air. Hence the patient rebreathes the air in the spirometer, removing more and more oxygen, but not rebreathing his exhaled CO_2. The second spirometer is the same except that there is no CO_2 absorber. The third contains 80-100% oxygen, and has no absorber. The subject now is connected to the first spirometer, and breathes from it for several minutes. At the end of this time, oxygen analysis of the spirometer air shows a marked reduction in the O_2 concentration, but the subject has not increased his respiratory rate or depth noticeably.

He now tries the second spirometer. Within a few minutes, he is puffing — and this occurs before there is much decrease in oxygen concentration. Results obtained with the third spirometer are essentially the same as with the second. From these observations it is concluded that accumulating CO_2 is a more effective stimulus to respiration than is decreasing oxygen concentration.

Summary

We have outlined the chief nervous and chemical means of regulating ventilation of the lungs, and described in some detail how each works. Others beyond the scope of this presentation have been deliberately ignored—not, we think, with any detriment to the therapist's understanding of the subject, as they are relatively of much less importance.

Attention has been drawn to the fact that elevated pCO_2 is a more potent stimulus to respiration under normal physiological conditions than is low oxygen tension.

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Acknowledgement: We wish to thank Mrs. Rosemary Whitacre for creating Figure 1 for this article. It is a composite from many sources.

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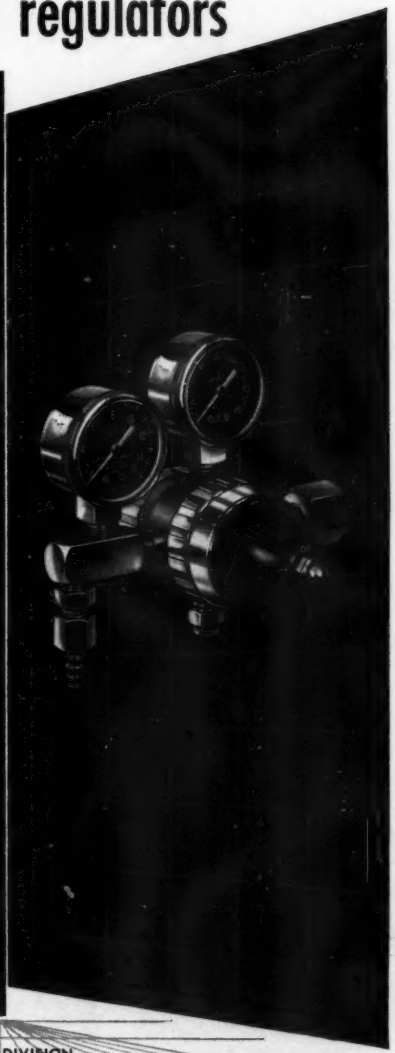

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The five young ladies standing, are receiving certificates as graduates of the Inhalation Therapy School established at Inter-Community Hospital at Covina, Calif. From left to right they are: Ingrid Persson, Lenora Brenton, Naomi Campbell, Betty Jo Keifer, and Rose Frederickson. Gentleman at far left, presenting the certificates is John Preston, Administrator of Inter-Community and seated at left is Constance Cypert, head of the Inhalation Therapy Department at the hospital and principal instructor. At her right is Helen Jenks, Director of Nurses. Mrs. Cypert is a member of the American Association of Inhalation Therapists and all five of her graduates have membership applications in process.

This group on the left is made up of students and staff of an Oxygen Therapy Course for nurses, orderlies and technicians given at Queen Mary Veterans Hospital in Montreal, Quebec. James Sharkey, a member of the Board of Directors of AAIT is senior Oxygen Therapist at the Hospital and was one of the instructors.

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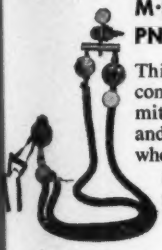

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FIRE HAZARDS IN OXYGEN TENTS

Static Sparks Not Hot Enough To Cause Tent Fires

The following are excerpts from correspondence between Dr. Alvan Barach and the Editor of the JAMA. We are sorry sufficient space is not available to print the letters in full.

We quote first (in full) the Minor Note in the June 30, 1956 JAMA which is responsible for the interchange:

"To the Editor: Please discuss the safety and practicability of an oxygent tent and the ordinary precautions observed in using it for a patient in a steel bed equipped with nylon sheets.—M.D., New York

"Answer:—Nylon in any form, sheets or bedclothes, on a steel bed, or any bed, under an oxygen tent is a fire hazard. A spark of static electricity could start a conflagration. Likewise, the following items are dangerous under an oxygen tent: wool blankets, electric heating pads, electric razors, call bells, flashlights, and, of course, anything burning, such as matches or cigarettes. Furthermore, only the least flammable substances should be allowed, e.g., talc instead of alcohol rubbing compound. Oil should never be used on valves or joints of apparatus."

The November 10, 1956 issue of JAMA carries a letter from Dr. Barach and Mr. Eckman, commenting on the foregoing. It says, in part:

"(The) answer to a query on "Hazards in Using Oxygen Tent" contains the extraordinary statement that a spark of static electricity could start a conflagration in an oxygen tent under certain circumstances such as the use of nylon in any form, sheet or bedclothes, as well as wool blankets. We do not believe there is any evidence to support such a contention. . . . We have never been able to start a fire with deliberately produced static sparks on wool blankets in atmospheres of 40 to 50% oxygen. . . . Certain anesthesia gases are inflammable and have a very low kindling or flash point; hence a static spark may ignite them very easily. In contrast, *oxygen is not inflammable;* it only supports combustion.

"For a fire to start in an oxygen atmosphere, it is necessary that the inflammable material in the oxygen atmosphere start burning. The kindling point of cotton sheets, wool or nylon blankets is considerably higher than that of the aforementioned anesthesia gases; hence it is much more difficult to ignite them. The presence of greater or lesser amounts of oxygen does not affect the kindling points of the materials.

"A static spark obtained from wool blankets or nylon uniforms has sufficient heat and intensity to raise the temperature of *explosive* gases above their kindling point; it cannot, however, do this with bed clothes, etc. Therefore, there is no danger of fire from this source in oxygen atmospheres.

"While *static* sparks do not create a hazard in oxygen tents, the presence of electrical appliances, any of which may 'short' or spark, does.

Alvan L. Barach, M.D.
Morris Eckman, B.S."

The Consultant (unidentified in the JAMA) who answered the original query

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in the June 30 issue, made reply to the above letter in the November 10 issue (in part) as follows:

"I agree with the writer that ordinarily a spark of static electricity would not cause a fire in an atmosphere of 40 to 50 percent oxygen. Experimentally one might attempt it a thousand times with negative results, but on the thousandth and one try have a positive result. One must not overlook the fact that patients expel intestinal gases and exhale anesthetic gases during recovery from anesthesia. There is always the possibility of low combustion point of foreign material having been spilled on bedding under the tent.

"The National Fire Protective Association's Standard for Nonflammable Medical Gas Systems, which has been adopted by the National Board of Fire Underwriters, specifically prohibits wool blankets and nylon fabrics within an oxygen tent."

We come now to the hitherto unpublished portion of the correspondence. October 10, 1956, Dr. Barach wrote Dr. J. F. Hammond, the Associate Editor of JAMA, some further comments on the subject, including the following statements from various authorities offered in support of his stand:

From Dr. George J. Thomas, Director, Dept. of Anesthesiology, St. Francis Hospital, Pittsburgh: "Due to the great interest of hospital people in the hazards of electrostatic charges produced by synthetic materials while receiving oxygen therapy, it was felt that laboratory investigation should be made to cover this subject. Following is part of the data that will be used in the paper we are preparing for publication in the near future.

"Woolen, nylon, silk, dacron and cotton fabrics were placed between electrodes of a specially designed instrument, and 30,000 volts of static electricity was made to pass through each sample for one to two minutes. A constant flow of oxygen was maintained throughout each test. The woolen material held the heat and could be ignited in 55 to 60 seconds when 30,000 volts of static electricity penetrated it. However, none of the other materials would ignite even after two minutes of a steady stream of 30,000 volts of static electricity in the presence of 95% oxygen. Laboratory tests were repeated several times with the same results.

"Because of the above findings, we concluded that static electricity offers no hazard in inhalation therapy."

Dr. Thomas brought out another important point in a subsequent communication which Dr. Barach also submitted to Dr. Hammond:

"I believe you can amplify the paragraph (about static versus electrical sparks) by stating that sparks in electrical equipment are of relatively longer duration as compared to static sparks, which transpire in less than a 10 millionth of a second. Moreover, the electric sparks may throw hot particles of metals against flammable solid materials in the presence of oxygen."

Dr. E. M. Papper, Chief of Anesthesia at Presbyterian Hospital (New York), had this to say: ". . . Dr. Thomas assures me that their experiments over the last 17 years show that 3 minutes after a patient is removed from an anesthesia machine, an inflammable mixture will no longer exist. Therefore the danger from static sparks with respect to anesthetic agents does not exist, providing anesthesia with inflammable agents has been discontinued 3 minutes before the use of the oxygen tent."

The final statement submitted by Dr. Barach was from the *pulmonary physiologist Morris Eckman* (now President of Respiration Aids, Inc.): "We know that hydrogen, an extremely explosive gas, is not hazardous until it reaches a concentration of 18% or more. If we assume that methane from the gut or anesthesia gases are four times as potent as explosive agents, it would be necessary to have a concentration of these gases of 4.5% or more.

A small oxygen tent canopy has an internal volume of approximately 25 cu. ft. or about 700 liters. Four and one half percent of 700 would be 30 liters. It would be impossible to obtain 30 liters of anesthesia gas and intestinal gas from a patient in order to raise the concentration of these gases to 4.5%."

As far as we know, JAMA has never published this rebuttal, and we feel that it contains rather important information—for inhalation therapists, at least. Whether the National Fire Protective Association has considered revising its stand in the matter or not is not known.

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ABSTRACTS

"Vital Capacity and Timed Vital Capacity in Men over 40," by Pemberton & Flanagan, in *J Appl Physiol* 3:291 (1956)

These researchers made a series of measurements on 428 men between 40 and 88 years of age, and on the basis of their findings, constructed a table of data in which Vital Capacity is correlated with height and weight. Using the nomogram, one may predict a man's vital capacity from his weight and height. The table is derived by use of a new formula devised by the authors, which is said to be more accurate than ones currently in common use.

"Alveolar CO_2 Levels in Acute Polio," by Crane et al, in *J Appl Physiol* 3:11 (1956)

This research team used a rapid infrared CO_2 analyzer to determine alveolar CO_2 concentrations in two series of polio patients. The first group of patients were not sufficiently below normal in their vital capacities to warrant use of tank respirators; those of the second group were.

Their findings indicate that the respirators evidently hyperventilated most of the patients from a normal alveolar pCO_2 value of 35-45 mm Hg down to about 25.

The tank respirators "seemed to create a demand for excessive ventilation, which could be alleviated by using other equipment."

"Antifoam Agents in Pulmonary Edema," by Reuben C. Balagot, M.D., Rosauro M. Reyes, M.D., and Max S. Sadove, M.D., in *JAMA* 163-630 Feb 23, 1957.

Evidently investigators as early as 1952 were trying silicones as antifoam agents to combat pulmonary edema, but only recently has this approach been tried more extensively. The present group induced pulmonary edema artificially in rabbits by intravenous injections of adrenaline, and then treated the condition with aerosols of ethyl alcohol (10 and 20%), octyl alcohol, and compound 5507, a new substance containing a silicone. They did not try 95% alcohol, as they found the vapors too irritating to the tracheobronchial tree.

They found the silicone agent more effective than ethyl alcohol, *and* without

Correction

In the June issue of Inhalation Therapy the abstract concerning "Anti-foam Agents" was printed with an incorrect final paragraph. With apologies to the authors, we are reprinting this abstract this month in its complete, correct form.

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the depressant action on the central nervous system produced by alcohol. It was tried with great success on a series of patients who had developed pulmonary edema from natural causes. It was administered with oxygen, via an IPPB machine, and proved successful.

"Alveolar CO_2 Measurements in Normal and Emphysematous Subjects," by James E. Kelsey, M.D., E. C. Oldham, M.S., and Steven Horvath, PhD., in *AMA Arch Int Med* 99:411 (March 1957)

These investigators contend that the emphysematous person's respiratory center is *not* depressed and insensitive to CO_2, and that, if anything, it is *more* sensitive to CO_2 than the normal person's. They say that the reason why, on breathing 7.5% CO_2 in O_2 the emphysematous person only doubles his minute volume, whereas the normal person triples his, is that the emphysematous person's rib cage is fixed and his lungs less elastic than those of normal individuals.

They arrived at this conclusion from studies in which they correlated results of standard pulmonary function tests (vital capacity, maximum ventilatory volume, etc.) with analyses of alveolar CO_2 concentration made before, during and after the tests.

The conclusions are provocative, and indicate the need for more research on this subject, as it challenges the position taken by standard texts.

"Structure of Respiratory (Terminal) Portion of Lungs," B. M. Fried, M.D. (New York City), in *A.M.A. Archives of Int Med* 98:691 (December 1956).

This article discusses other functions of lungs. They appear to take up fats in the same way they phagocytose (devour) bacilli. Evidently also detoxify alkaloids (chiefly by oxidation), and engage in glycolysis, absorption of water, etc. These functions are all performed by epithelial cells lining the terminal air sacs (alveoli).

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